Amendments to the Specification:

Please amend the specification as follows:

Please replace the Title on page 1 with the following:

--Compositions of Angiopoietin, fragments, mutants and analogs thereof and uses of the same--

Please replace the paragraph on page 27, line 14, of the specification with the following:

--Two Ang-I mutants have been established in which either the linker peptide region of Ang-I ($_{258}$VHNLVSL$_{265}$CTKEGVLLKGGKREEEKPF$_{283}$) (SEQ ID NO. 37) was deleted (Ang-$1_{minuslinker}$) or the Cys265 residue in the region was mutated to Ser (Ang-$1_{cys265ser}$).--

Please replace the paragraph on page 28, line 19, of the specification with the following:

--A peptide, L$_{265}$CTKEGVLLKGGKREEEKPF$_{283}$ (SEQ ID NO. 38), derived from the linker peptide region was found to inhibit the incorporation of Ang-1 proteins to the ECM in cell culture condition. This result suggests that the linker peptide or its derivatives (peptides and small molecules) can potentially be used to modulate the ECM binding of Ang-1, therefore the bioactivity and availability of Ang-1.--

Please replace the paragraph on page 35, line 20, of the specification with the following:

--It has been shown recently that the linker peptide region between the coiled-coil and the fibrinogen-homology domain (FHD) of Ang-1 likely mediates that interaction between Ang-1 and the ECM (32). The linker peptide region contains 26 amino acids ($_{258}$VHNLVSL$_{265}$CTKEGVLLKGGKREEEKTIF$_{283}$) (SEQ ID NO. 39), and is highly conserved among different species. There is 96% identity at the amino acid level between human and mouse in this region. To confirm that the linker peptide region mediates the ECM binding of Ang-1 and determine the role of the cysteine265 residue, which is conserved among different

species and unique to Ang-1, in the ECM binding of Ang-1, two Ang-1 mutants, in which either the linker peptide region was deleted (Ang-$1_{minuslinker}$), or the cysteine265 residue was mutated into a serine residue (Ang-1cys265ser) were generated. The expression constructs (pEF/6His-v5, Invitrogen) containing wild type Ang-1, Ang-$1_{minuslinker}$, and Ang-1cys265ser were used to transfect Cos-7 cells using Lipofectamine™ liposome reagent. Seventy-two hours after the transfection, the cell culture supernatants and the ECM materials deposited by the transfected cells were analyzed by Western blotting with anti-v5 mAb to determine the distribution of Ang-1 and the Ang-1 mutants. The results showed that the Ang-1 mutant that lacks the linker peptide region, Ang-$1_{minuslinker}$, displayed dramatically reduced binding to the ECM, and the mutation of $cysteine_{265}$ residue to a serine reduced the ECM binding of the mutants. Both mutations alter the aggregation pattern as well compared to that of wild type Ang-1 (Fig 2). This result demonstrated the importance of the linker peptide region and the cysteine265 residue in the ECM binding of Ang-1 and provided us with useful tools to study how the ECM binding affects the function of Ang-1.--